167 Epidemic outbreak of *Burkholderia cepacia* in a reference center of cystic fibrosis in Buenos Aires, Argentina

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Introduction: The prevalence of *Burkholderia cepacia* complex (BCC) has increased in many centers of the world. Infection control is one of the main tools for prevention.
Objective: To describe strategies adopted in an epidemic outbreak of BCC.
Materials and Methods: Descriptive analysis. We review clinical histories from January 2004 to July 2007 of 248 patients (pts) with Cystic Fibrosis (CF) assisted in our Center.
Results: BCC were isolated in 64 pts (35 in 2004, 19 in 2005, 9 in 2006 and 1 in 2007). It was isolated in only one opportunity in 36 pts, twice in 19 and three or more times in 9. Of the 64 pts, 46 continue in follow-up, and eight of them died, none of cepacia syndrome or of worsening attributed to BCC. The genotype analysis made in 19 children in an international laboratory of reference confirmed BCC in 17 with the same strain and in 2 *Delftia tsuruhatensis*. After the BCC's first isolations we intensified infection control policies already established. During 2005 and 2006 the concurrence of patients to the Center was suspended, and the professionals of the team went to the patients' homes and other hospitals of the state of Buenos Aires for their assistance. In 2007 the decentralization was kept on 5 areas of assistance (4 far from the hospital) according bacteriology. The incidence experienced a significant decrease.
Conclusion: The segregation of children with BCC and the infection control policies are standardized and their benefits have been demonstrated. The efforts of the professional team must be recognized. The psychological impact which patients experience is an important obstacle that should be minimized.

168* Invasion of *Burkholderia cepacia* complex isolates into lung epithelial cells involves glycolipid receptors

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B. cepacia is a Gram-negative bacteria comprising at least nine species and collectively referred to as the *B. cepacia* complex (Bcc). Although the prevalence of this opportunistic pathogen has declined due to stringent isolation procedures, when it does colonise an individual with cystic fibrosis, it remains a major cause of morbidity and mortality. *B. cenocepacia* and *B. multivorans* are the most virulent and clinically relevant species. One of the mechanisms of pathogenesis of Bcc is invasion of lung epithelial cells, with the two most virulent species invading most readily. Prior to invasion, these bacteria need to bind to the lung epithelial cells. Previously, it has been shown that Bcc bind to many glycolipid receptors, including gangliosides and globosides, on lung epithelia. We have now demonstrated that these glycolipid receptors are essential for invasion. Enzyme treatment to remove galactose moieties from the lung cell surface dramatically inhibited invasion, while removal of sialic acid did not. In addition, treatment of lung cells with sphingolipid biosynthetic pathway inhibitors at non-cytotoxic levels also significantly reduced invasion by Bcc strains. In contrast, results obtained following treatment with N-acetylcysteine, indicated that mucus did not play a major role in the invasion process. Using labelled bacteria, we found that different strains within Bcc showed different preferential binding to particular glycosphingolipids. We are now evaluating a series of novel glycoconjugate molecules for their ability to inhibit bacterial binding and invasion and thereby reduce both colonisation and virulence potential of this difficult CF pathogen.
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169 Degradation of host defence molecules by CF-related pathogens grown as biofilms

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We investigated the ability of secreted bacterial proteinases from three pathogens [*Burkholderia multivorans* (*Bm*), *Burkholderia cenocepacia* (*Bc*), and *Pseudomonas aeruginosa* (*Pa*)] involved in chronic bacterial infections in cystic fibrosis to degrade various host defence-related molecules. These included two endogenous proteinase inhibitors, secretory leukocyte proteinase inhibitor (rhSLPI) and alpha-1 antitrypsin (AAT); two relevant immunoglobulins, secretory IgA (sIgA) and IgG, and two proteins important in innate immunity, lactoferrin and lysozyme.
Host defence-related molecules were co-incubated with cell-free supernatants from 48 hour biofilm cultures, grown on mucin-coated microplates, from all three pathogens under investigation (six isolates from each species). No degradation of AAT, sIgA, IgG, and lactoferrin was observed for any of the organisms. Of the 18 isolates tested, only one demonstrated the ability to degrade lysozyme. This was an environmental isolate of *Pa* which was included as a comparison for the predominantly clinically relevant isolates used in the study.
In contrast, all isolates of *Bm* (n = 4) and *Pa* (n = 4) were able to degrade rhSLPI however, out of five bacterial isolates tested for *Bc* only two demonstrated a limited ability to degrade the molecule with >95% of the protein band still remaining intact at the end of the experiment.
This study demonstrates that the majority of the host defence molecules investigated are resistant to degradation by bacterial proteinases from *Bm*, *Bc* and *Pa* when grown as a biofilm. However, rhSLPI was vulnerable to significant degradation which could result in aberrant serine proteolysis in regions of the lungs containing biofilm growth.

170 Cross-infection by *Staphylococcus aureus* among cystic fibrosis patients

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Chronic infection and progressive pulmonary destruction, in Cystic Fibrosis (CF) patients, are mainly caused by *Pseudomonas aeruginosa* and *Staphylococcus aureus*. *S. aureus* is generally the first infectious agent. These infections may be acquired from people without CF or from cross-infection among patients.
In this study the authors intended to identify a possible cross-infection by *S. aureus* among patients followed at the CF Centre of Hospital Santa Maria (paediatric group: 68 pts). These patients are already divided into cohorts, in order to minimize cross-infection by *P. aeruginosa*, *Burkholderia cepacia* and MRSA.
Isolates of *S. aureus* identified on microbiological exams of sputum or respiratory secretions, between May 2005 and April 2006, have been studied (144 isolates from 44 patients).
Isolates have been characterized by antibiotic susceptibility tests and pulsed-field gel electrophoresis (PFGE) and the restriction patterns compared by the Bionumerics computer program. Twenty different clones were identified. Five of these clones comprised 65% of the 144 isolates and 25% belonged to the major one.
A tendency for grouping the clones by cohort of patients has been found (Wallace coefficient 15.3%, confidence interval from 13.9 to 34.9%, not containing the expected value of 12.9% in the case of independence).
These results suggest that there is cross-infection by *S. aureus* among patients with CF.
No relationship, with statistical significance, was found between clinical parameters of disease severity (body mass index, respiratory function tests and hospital admissions) and any specific clone (Kruskal-Wallis test).
This study points the necessity to cohort separately patients infected or not with *S. aureus*.